

## WEST Search History

DATE: Saturday, January 04, 2003

<u>Set Name</u>	<u>Query</u>	<u>Hit Count</u>	<u>Set Name</u>
side by side			result set
	<i>DB=USPT; PLUR=YES; OP=OR</i>		
L7	(4507466)[pn]	1	L7
L6	dendrimer same ( Cys or cysteine)	18	L6
L5	dendrimer near cysteine	0	L5
L4	dendrimer near alanine	0	L4
L3	dendrimer	878	L3
L2	e9 adj starburst	3	L2
L1	(4766106)[PN] OR (4847325)[PN]	2	L1

END OF SEARCH HISTORY

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 12:35:05 ON 04 JAN 2003

L1 11034 S WONG H?/AU OR RHODE P?/AU OR WEIDANZ J?/AU OR GRAMMER S?/AU O  
L2 0 S L1 AND ((MULTIMER OR MULTIVALENT) AND MHC)  
L3 14 S L1 AND (FUSION AND MHC)  
L4 10 DUP REM L3 (4 DUPLICATES REMOVED)  
L5 13 S (SOLUBLE (10N) (CLASS (1N) II) (10N) FUSION) AND MHC  
L6 11 DUP REM L5 (2 DUPLICATES REMOVED)  
L7 10 S L6 NOT L4  
L8 0 S L5 AND DENDRIMER?  
L9 7 S DENDRIMER? AND MHC  
L10 3 DUP REM L9 (4 DUPLICATES REMOVED)

=> s 3dt52.5

6 3DT52  
1759720 5

L1 6 3DT52.5  
(3DT52(W) 5)

=> dup rem l1

PROCESSING COMPLETED FOR L1

L2 6 DUP REM L1 (0 DUPLICATES REMOVED)

=> dis l2 1-6 ibib abs

L2 ANSWER 1 OF 6 MEDLINE

ACCESSION NUMBER: 1998026161 MEDLINE

DOCUMENT NUMBER: 98026161 PubMed ID: 9379039

TITLE: Rigidification of the alpha2 helix of an MHC class I molecule by a valine to proline mutation in position 165 does not prevent peptide-specific antigen presentation.

AUTHOR: Plaksin D; Polakova K; Mage M G; Margulies D H

CORPORATE SOURCE: Laboratory of Immunology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, MD 20892-1892, USA.

SOURCE: JOURNAL OF IMMUNOLOGY, (1997 Nov 1) 159 (9) 4408-14.  
Journal code: 2985117R. ISSN: 0022-1767.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199711

ENTRY DATE: Entered STN: 19971224

Last Updated on STN: 19971224

Entered Medline: 19971112

AB Although classical MHC class I glycoproteins bind peptide Ags for display at the cell surface, some MHC class I-related molecules such as the neonatal Fc receptor (FcRn) execute their function without binding peptide ligands. The three-dimensional structure of the FcRn suggested that a substitution of the conserved valine at position 165 of the alpha2 helix by proline contributed to a kink in the position of this helix relative to the alpha1 helix, and resulted in closing of the potential peptide-binding cleft. To test the contribution of proline 165 to the occlusion of the cleft and the binding of potential antigenic peptides, we introduced this mutation into the classical murine MHC class I molecule, H-2Dd, and characterized the ability of such a mutant to present peptide Ags to either a peptide-specific, H-2Dd-restricted T cell hybridoma (B4.2.3), or an allospecific, peptide-dependent, T cell hybridoma (3DT52.5.8). We show that the V165P mutation, expressed at the cell surface either in H-2Dd or in a single chain membrane version of H-2Dd, fails to eliminate recognition of the peptide/MHC complexes by two different T cells. Evaluation of a panel of synthetic substituted peptides suggests that subtle differences in the fine specificity of presentation can be discerned. Thus, the proline substitution at position 165 of FcRn and some other class I-like molecules is not the sole cause of the lack of peptide presentation.

L2 ANSWER 2 OF 6 MEDLINE

ACCESSION NUMBER: 96173773 MEDLINE

DOCUMENT NUMBER: 96173773 PubMed ID: 8596036

TITLE: Mutations in human CD4 impair the functional interaction with different human and mouse class II isotypes and alleles.

AUTHOR: Fleury S; Huang B; Zerbib A; Croteau G; Long E O; Sekaly R P

CORPORATE SOURCE: Immunology Laboratory, Montreal Clinical Research

SOURCE: Institute, Canada.  
 JOURNAL OF IMMUNOLOGY, (1996 Mar 1) 156 (5) 1848-55.  
 Journal code: 2985117R. ISSN: 0022-1767.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
 ENTRY MONTH: 199604  
 ENTRY DATE: Entered STN: 19960424  
 Last Updated on STN: 19960424  
 Entered Medline: 19960415

AB The structure-function of the CD4-class II MHC interaction was investigated. Two functional assays were used to assess the responses of the 3DT52.5.8 murine T cell hybridoma expressing human CD4 (h-CD4) or murine CD4 (m-CD4). First, we determined the responses of the CD4+ and CD4-effector cells toward DAP-3 cells co-expressing the cognate alloantigen H-2Dd together with several human (DRw52b, DR4-Dw4, DR2A, and DPw2) and murine (I-Ab, I-Ak, IA alpha b I-A beta k and I-Ek) class II alleles and isotypes. We found that h-CD4 and m-CD4 strongly enhance the T cell response to H-2Dd, demonstrating that interspecies CD4/class II interactions occur efficiently. Furthermore, mutations in h-CD4 at positions 19, 89, and 165 markedly reduced the interaction with both human class II and mouse class II, indicating that the structural features of this cross-species interaction are strongly conserved. This was further supported by the finding that a h-CD4 deletion mutant (deletion F43-S49) interacted with both human and murine class II. Moreover, as 3DT cells express the responsive V beta element for the bacterial superantigen staphylococcal enterotoxin B, a co-receptor assay was conducted. DAP-3 cells expressing only class II molecules were used as APCs to present staphylococcal enterotoxin B to h-CD4+ and m-CD4+ T cells. h-CD4 and m-CD4 were able to enhance the T cell response to staphylococcal enterotoxin B, further demonstrating the conservation of the CD4-class II MHC interaction.

L2 ANSWER 3 OF 6 MEDLINE

ACCESSION NUMBER: 87215948 MEDLINE  
 DOCUMENT NUMBER: 87215948 PubMed ID: 3034434  
 TITLE: The role of the L3T4 molecule in mitogen and antigen-activated signal transduction.  
 AUTHOR: Rosoff P M; Burakoff S J; Greenstein J L  
 CONTRACT NUMBER: AM07715 (NIADDK)  
 AM31405 (NIADDK)  
 DK34605 (NIDDK)

SOURCE: CELL, (1987 Jun 19) 49 (6) 845-53.  
 Journal code: 0413066. ISSN: 0092-8674.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198707  
 ENTRY DATE: Entered STN: 19900303  
 Last Updated on STN: 19970203  
 Entered Medline: 19870723

AB We investigated the role of the L3T4 molecule in mitogen and antigen-initiated signal transduction in the L3T4(+) murine T cell hybridoma, 3DT52.5.9 and an L3T4(-) variant, 3DT52.5.24. Both Concanavalin A (Con A) and specific antigen stimulated increases in cytosolic-free calcium ([Ca2+]i), phosphatidylinositol turnover, and interleukin-2 (IL-2) production in both cell lines. About 85% of the stimulated rise in [Ca2+]i was from an extracellular source. Anti-L3T4 monoclonal antibody (MAb) inhibited 90% of antigen- and 50% of Con A-stimulated increases in [Ca2+]i and IL-2 production but had no effect on the ability of either activation signal to stimulate phosphatidylinositol turnover in the parent L3T4(+) cells.

Stimulus-response coupling in the L3T4(-) cells was unaffected by the MAb. The anti-L3T4-insensitive increase in [Ca<sup>2+</sup>]<sub>i</sub> induced by Con A was inhibited by EGTA, suggesting that this mitogen also stimulated an influx of Ca<sup>2+</sup> via an additional transport mechanism distinct from that stimulated by antigen. The fact that anti-L3T4 antibodies inhibit antigen and Con A-stimulated Ca<sup>2+</sup> transport and IL-2 production without affecting phosphatidylinositol turnover suggests that L3T4 may play a critical role in modulating the activation of the T cell receptor-associated Ca<sup>2+</sup> transporter in T cell stimulus-response coupling.

L2 ANSWER 4 OF 6 MEDLINE

ACCESSION NUMBER: 85236163 MEDLINE  
 DOCUMENT NUMBER: 85236163 PubMed ID: 3925069  
 TITLE: Role of L3T4 in antigen-driven activation of a class I-specific T cell hybridoma.  
 AUTHOR: Greenstein J L; Malissen B; Burakoff S J  
 CONTRACT NUMBER: AI-17258 (NIAID)  
 CA-07324 (NCI)  
 SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1985 Jul 1) 162 (1) 369-74.  
 Journal code: 2985109R. ISSN: 0022-1007.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198508  
 ENTRY DATE: Entered STN: 19900320  
 Last Updated on STN: 19980206  
 Entered Medline: 19850815

AB The expression of L3T4/Lyt-2 on murine T cells has led to the association of these surface markers with recognition of either class II or class I major histo-compatibility complex (MHC) antigens. It has been suggested that these T cell surface antigens interact with nonpolymorphic determinants on MHC antigens. We have examined the role of L3T4 in the recognition of H-2Dd by the T cell hybridoma, **3DT52.5**. Mouse L cells transfected with either the H-2Dd gene, or with both the alpha and beta genes of I-Ak and the H-2Dd gene have been used to assess the role of an L3T4/Ia interaction at varying doses of H-2Dd. A role of L3T4 in activation of **3DT52.5** becomes evident only at limiting doses of antigen. It appears that an L3T4/Ia interaction can influence T cell function during suboptimal stimulation, implying that the L3T4/Ia interaction serves to raise the functional affinity of interaction between the T cell and the antigen-bearing cell.

L2 ANSWER 5 OF 6 MEDLINE

ACCESSION NUMBER: 84163773 MEDLINE  
 DOCUMENT NUMBER: 84163773 PubMed ID: 6200564  
 TITLE: The role of L3T4 in recognition of Ia by a cytotoxic, H-2Dd-specific T cell hybridoma.  
 AUTHOR: Greenstein J L; Kappler J; Marrack P; Burakoff S J  
 CONTRACT NUMBER: AI-17258 (NIAID)  
 AI-18785 (NIAID)  
 CA-07324 (NCI)  
 SOURCE: JOURNAL OF EXPERIMENTAL MEDICINE, (1984 Apr 1) 159 (4) 1213-24.  
 Journal code: 2985109R. ISSN: 0022-1007.  
 PUB. COUNTRY: United States  
 DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
 LANGUAGE: English  
 FILE SEGMENT: Priority Journals  
 ENTRY MONTH: 198405  
 ENTRY DATE: Entered STN: 19900319  
 Last Updated on STN: 19980206  
 Entered Medline: 19840514

AB The expression of T4/T8 surface markers on human T cells and of L3T4/Lyt-2 on murine T cells has lead to the association of these surface markers with recognition of either class II or class I major histocompatibility complex (MHC) antigens. It has been suggested that these T cell surface antigens interact with MHC antigens. We have examined the role of L3T4 in the recognition of Dd by the T cell hybridoma, **3DT52.5**. This T cell hybridoma was found to be specific for the N/C1 domain of Dd. The recognition of a class I antigen by an Lyt-2-, L3T4+ T cell hybridoma allowed the separate evaluation of interactions between L3T4/Ia and the T cell antigen receptor, Dd. Recognition by this hybridoma resulted in the production of interleukin 2 (IL-2) and cytolytic activity. Antibody blocking experiments have demonstrated that L3T4 was involved in triggering the effector function of **3DT52.5** only on Ia+ stimulator or target cells. We have demonstrated that an L3T4+, Dd-specific T cell hybridoma, **3DT52.5**, uses the L3T4 molecule to directly interact with nonpolymorphic Ia determinants.

L2 ANSWER 6 OF 6 MEDLINE

ACCESSION NUMBER: 84009070 MEDLINE  
DOCUMENT NUMBER: 84009070 PubMed ID: 6604749  
TITLE: An IL 2-secreting T cell hybridoma that responds to a self class I histocompatibility antigen in the H-2D region.  
AUTHOR: Endres R O; Marrack P; Kappler J W  
CONTRACT NUMBER: AI-18785 (NIAID)  
SOURCE: JOURNAL OF IMMUNOLOGY, (1983 Oct) 131 (4) 1656-62.  
Journal code: 2985117R. ISSN: 0022-1767.  
PUB. COUNTRY: United States  
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)  
LANGUAGE: English  
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals  
ENTRY MONTH: 198311  
ENTRY DATE: Entered STN: 19900319  
Last Updated on STN: 19970203  
Entered Medline: 19831123

AB A self-reactive T cell hybridoma that secretes IL-2 in response to H-2d haplotype cells resulted from a fusion of BALB/cBy lymph node cells with the AKR thymoma BW5147. The lymph node cells used had been enriched for cells reactive to (TG)-A--L, but neither this antigen nor fetal calf serum were required for stimulation of the hybridoma designated **3DT52.5**. The gene product responsible for stimulation mapped to the H-2D region. Allogeneic cells of the b, f, k, q, and s haplotypes failed to stimulate. Not all H-2d haplotype cells were effective stimulators of **3DT52.5**. Peritoneal cells and splenic B cells were much more stimulatory than splenic T cells. Most tumor cell lines of H-2d derivation and of B cell or macrophage/monocyte lineage were stimulatory, whereas H-2d T cell lines were not. The capacity to stimulate **3DT52.5** did not correlate with the ability to stimulate I region-restricted hybridomas, or with the ability to be induced to stimulate such hybridomas. Stimulatory cell lines did not apparently produce a soluble factor required for stimulation, and negative cell lines were not inhibitory. The monoclonal antibody 27-11-13, which reacts with H-2D of the b, d, and q haplotypes, inhibited stimulation of **3DT52.5** but did not inhibit stimulation of the sibling hybridoma **3DT18.11**, which responds to (TG)-A--L plus I-Ad. Conversely, the monoclonal anti-I-Ad antibody MK-D6 inhibited stimulation of **3DT18.11** but not **3DT52.5**. Although it is clear that **3DT52.5** recognizes a class I antigen coded for in the H-2D region, the precise molecular nature of the antigen is unknown. The structure of the antigen receptor on this hybridoma may prove to be of interest when it can be compared with receptors found on T cell hybridomas restricted by class II histocompatibility antigens.